Product Data Sheet

Product Name: DQP 1105
Cat. No.: GC10085

Chemical Properties

Cas. No.	380560-89-4		
Chemical Name	4-((R)-5-(4-bromophenyl)-3-((R)-6-methyl-2-oxo-4-phenyl-2,3-dihydroquinolin-3-yl)-4,5-dihydro-1H-pyrazol-1-yl)-4-oxobutanoic acid		
SMILES	BrC1=CC=C(C=C1)[C@@H]2N(C(CCC(O)=O)=O)N=C([C@H]3C(C4=CC=CC=C4)=C(C=C(C)C=C5)C5=NC3=O)C2		
Formula	$C_{29}H_{24}BrN_3O_4$	M.Wt	558.42
Solubility	<55.84mg/ml in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	DQP 1105		

Protocol

Cell experiment [1]:

Cell lines	Mouse primary astrocytes
Preparation Method	Mouse primary astrocytes were cultured in high-glucose DMEM medium supplemented with 10% fetal bovine serum (FBS) and 1% Penicillin/Streptomycin at 37℃ in the presence of 5% CO_2. The anti-AQP4 IgG (5μg/ml) and human complement C3 (10μg/ml) were added into the culture medium of astrocytes for 24h to induce the neuromyelitis optica phenotype. After treating with 20μM DQP 1105 for 24 hours, the levels of Cx43 protein and VIP protein were analyzed.
Reaction Conditions	20μM; 24h
Applications	DQP 1105 treatment reduced the levels of Cx43 protein and VIP protein in astrocytes.

Animal experiment [2]:

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Animal models	CD-1 mice
Preparation Method	3-month-old male CD1 mice (35-48g) were maintained in a temperature and humidity-controlled environment under a 12-12h dark-light cycle (06:00 to 18:00 hours for light) with food and water available ad libitum. A single intraperitoneal injection of 90mg/kg dose of DQP 1105 or ketamine (20mg/kg) as a positive control was administered, and a rotating rod test was conducted within 30 minutes.
Dosage form	90mg/kg for once; i.p.
Applications	DQP 1105 treatment significantly impaired motor coordination in mice.

References:
[1] Xue H, Wu M, Wang Y, et al. The circadian rhythms regulated by Cx43-signaling in the pathogenesis of Neuromyelitis Optica[J]. Frontiers in Immunology, 2023, 13: 1021703.
[2] Pálfi E, Lévay G, Czurkó A, et al. Acute blockade of NR2C/D subunit-containing N-methyl-D-aspartate receptors modifies sleep and neural oscillations in mice[J]. Journal of Sleep Research, 2021, 30(4): e13257.

Background

DQP 1105 is a noncompetitive antagonist of N-methyl-D-aspartate (NMDA) receptor, which inhibits GluN2C- and GluN2D-containing receptors with IC_{50} values of 7.0 and 2.7μM [1]. DQP 1105 can reversibly weaken the membrane potential and circadian rhythm of clock gene expression in suprachiasmatic nucleus (SCN) neurons by inhibiting NMDAR2C[2]. DQP 1105 has been widely used for regulating neuronal synaptic responses[3].

In vitro, DQP 1105 treatment (20μM) for 24 hours significantly reduced the levels of Cx43 protein and VIP

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protein in astrocytes, as well as the level of Clock mRNA[4]. 15μM DQP 1105 treatment for 10 days significantly promoted the growth of pyramidal cells and increased the number of apical dendritic branches of these cells[5]. Treatment with 10μM DQP 1105 for 4 hours can reduce the expression of p-PI3K and p-Akt in CTX-TNA2 cells treated with lipopolysaccharide (LPS) [6].

In vivo, DQP 1105 treatment via a single intraperitoneal injection at a dose of 90mg/kg impaired the motor coordination ability of CD-1 mice within 30 minutes[7]. A single intraperitoneal injection of 28mg/kg dose of DQP 1105 reduced the frequency of epileptic seizures in mice within 2 hours[8].

References:

[1] Acker T M, Yuan H, Hansen K B, et al. Mechanism for noncompetitive inhibition by novel GluN2C/D N-methyl-D-aspartate receptor subunit-selective modulators[J]. Molecular pharmacology, 2011, 80(5): 782-795.
[2] Brancaccio M, Edwards M D, Patton A P, et al. Cell-autonomous clock of astrocytes drives circadian behavior in mammals[J]. Science, 2019, 363(6423): 187-192.
[3] Mahmoud H, Martin N, Hildebrand M E. Conserved contributions of NMDA receptor subtypes to synaptic responses in lamina II spinal neurons across early postnatal development[J]. Molecular brain, 2020, 13(1): 31.
[4] Xue H, Wu M, Wang Y, et al. The circadian rhythms regulated by Cx43-signaling in the pathogenesis of Neuromyelitis Optica[J]. Frontiers in Immunology, 2023, 13: 1021703.
[5] Köhler I, Rennau L M, Hoffmann L, et al. Activation of GluN2D-containing NMDA receptors promotes development of axons and axon-carrying dendrites of cortical interneurons[J]. Cerebral Cortex, 2025, 35(6): bhaf136.
[6] Gao R, Ali T, Liu Z, et al. NMDAR (2C) deletion in astrocytes relieved LPS-induced neuroinflammation and depression[J]. International Immunopharmacology, 2024, 132: 111964.
[7] Pálfi E, Lévay G, Czurkó A, et al. Acute blockade of NR2C/D subunit-containing N-methyl-D-aspartate receptors modifies sleep and neural oscillations in mice[J]. Journal of Sleep Research, 2021, 30(4): e13257.
[8] Lozovaya N, Gataullina S, Tsintsadze T, et al. Selective suppression of excessive GluN2C expression rescues early epilepsy in a tuberous sclerosis murine model[J]. Nature communications, 2014, 5(1): 4563.

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